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Variation in virus effects on host plant phenotypes and insect vector behavior: what can it teach us about virus evolution?

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Virus infection can elicit changes in host plant cues that mediate vector orientation, feeding, and dispersal. Given the importance of plant cues for vector-mediated virus transmission, it is unlikely that selection is blind to these effects. Indeed, there are many examples of viruses altering plant cues in ways that should enhance transmission. However, there are also examples of viruses inducing transmission-limiting plant phenotypes. These apparently mal-adaptive effects occur when viruses experience host plant environments that also limit infectivity or within-host multiplication. The apparent link between virus effects and pathology argues for consideration of prior evolutionary relationships between viruses and host plants in order to understand how viruses might evolve to manipulate vector behavior via effects on host plant cues.

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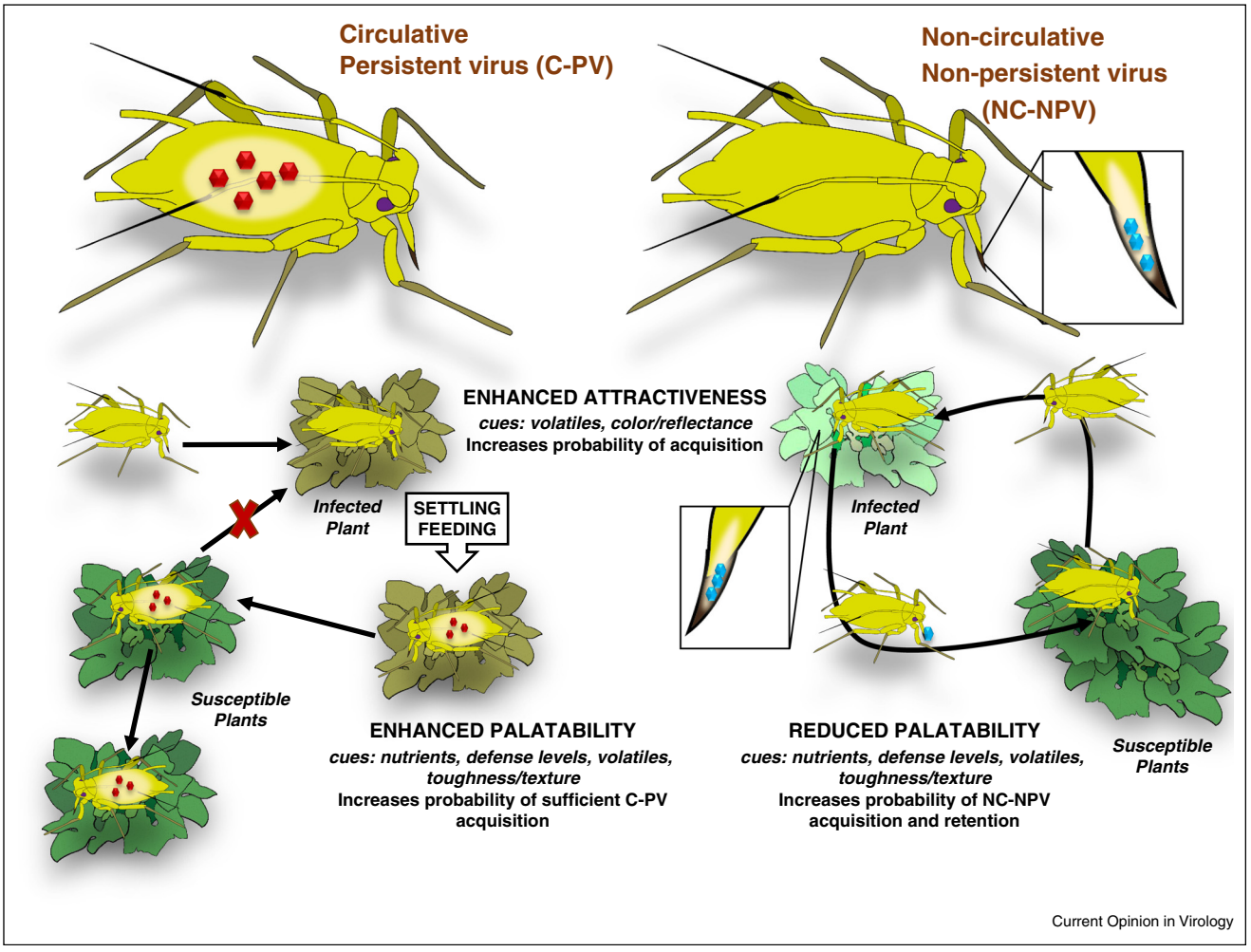
Introduction

Virus infection can fundamentally alter the way that host plants interact with other organisms. In particular, many viruses change host plant phenotypes in ways that influence interactions with vectors [1–3,4•], with significant implications for virus transmission. These phenotypic changes involve alteration of visual or odor cues mediating vector orientation to plants (recruitment), quality or palatability cues mediating feeding behavior (virus acquisition), and effects on vector movement from infected to susceptible hosts (virus transmission) [3,4•]. In the case of viruses that also circulate and/or replicate in their vectors, phenotype changes that permit discrimination between infected and healthy plants can also interact with direct effects of the virus on vector physiology and behavior [5–9]. For example, virus-free aphids prefer to settle and feed on wheat infected with *Barley yellow dwarf virus* (BYDV), but once aphids have acquired the virus, their settling preferences change to favor healthy plants, facilitating virus spread [5]. This example, along with other recent studies reporting complex, transmission-conducive effects of viruses on plants and vectors [4•,10], support the hypothesis that such effects are the result of specific viral adaptations, and are not just by-products of pathology. However, much of this work has ignored natural genetic variation in virus and host plant populations. Among insect-vectored viruses, genetic diversity is generated by mutations that occur during replication (particularly for RNA viruses) coupled with biotic and abiotic heterogeneity in plant communities [11–14], and variation in vector competencies or host preferences [15]. Despite this diversity, most studies to date involve cultivated model host plants infected with virus strains originally isolated from monocultures and subsequently maintained in the laboratory [3,4•]. A more robust test of the adaptive significance of virus effects on plant phenotypes would be one that considers natural genetic variation in both the virus and the host plant, as well as the ecological context in which different virus isolates have evolved. This review highlights examples of recent progress toward this goal and synthesizes this work to gain insight into the factors shaping the evolution of virus effects on plant cues mediating plant–vector interactions.

Expectations for virus effects on host plant phenotypes

Since the earliest reports of viruses influencing vectors via a shared host plant [16], there has been speculation about whether these effects constitute evidence of specific virus adaptations for manipulating plant phenotypes in ways that enhance transmission. Unlike clear cases of manipulation involving higher organisms (reviewed in [17,18]), for plant viruses it is often difficult to distinguish adaptive effects from by-products of infection because viruses alter suites of existing cues, such as volatile emissions or free amino acids [19,20], rather than inducing complex morphological [21•] or behavioral [18] changes. Nonetheless, given the importance of host cues for vectors, selection should tend to favor virus genotypes that alter plant phenotypes in ways that are generally conducive to transmission (no effect or a positive change) and disfavor virus genotypes that change plant phenotypes in ways that have clear negative effects on transmission [3,4•].

Figure 1



Expectations for transmission-mechanism specific effects of viruses on host plant phenotypes. Plant viruses can be either circulative or non-circulative. Circulative viruses are acquired during long-term feeding, usually in the phloem, after which they circulate within the vector (sometimes replicating) and migrate to specific tissues, such as salivary glands, from which they can be inoculated to multiple plants. Since long bouts of feeding are required for circulative virus acquisition and inoculation, it is expected that these viruses should have neutral to positive effects on plant attractiveness (to encourage vector contacts) and palatability or quality (to ensure uptake of a sufficient number of virions). Following virion acquisition, it is beneficial for the virus if the vector disperses from the infected plant and then exhibits a preference for healthy plants (as for C-PVs, shown above). In contrast, acquisition of most non-circulative viruses (particularly NC-NPVs, shown above) is favored by vectors making brief probes of non-vascular epidermal cells, then rapidly dispersing from infected plants to healthy plants. Non-circulative viruses are not retained internally, instead binding to specific regions of the mouthparts (e.g. NC-NPVs that adhere to aphid stylets, shown above) or foregut (most semi-persistently transmitted viruses, not depicted) for a few hours to a few days. This transmission mechanism should be facilitated by phenotypic changes to hosts that render them attractive to vectors, but less palatable following acquisition of plant cues and virions in order to encourage the rapid dispersal necessary for transmission. It is beneficial for a non-circulative virus if this shift in preference is temporary, as this will ensure that vectors do not permanently avoid infected hosts. For comprehensive reviews on each transmission mechanism, see [65,66].

One key line of evidence supporting the hypothesis that virus effects on plant cues are not mere by-products of infection is the apparent convergence of phenotypic effects across distantly-related pathogens transmitted in the same way [3,4•]. Viruses sharing a transmission mechanism will benefit from similar sequences of vector orientation, feeding, and dispersal behavior, and are thus expected to induce similar phenotypic shifts in host plants (Figure 1). This hypothesis has been recently discussed in two reviews [3,4•] which document clear patterns of congruency in virus effects based on shared transmission mechanism. But despite this broad pattern, individual reports of apparently mal-adaptive effects also exist (Table 1). This seems unlikely to be the result of publication bias, since mal-adaptive effects of a virus on its own transmission are still of ecological interest [22]. Rather, these reports may constitute evidence that viruses experience trade-offs in their ability to alter the phenotypes of multiple plant genotypes or species.

Table 1

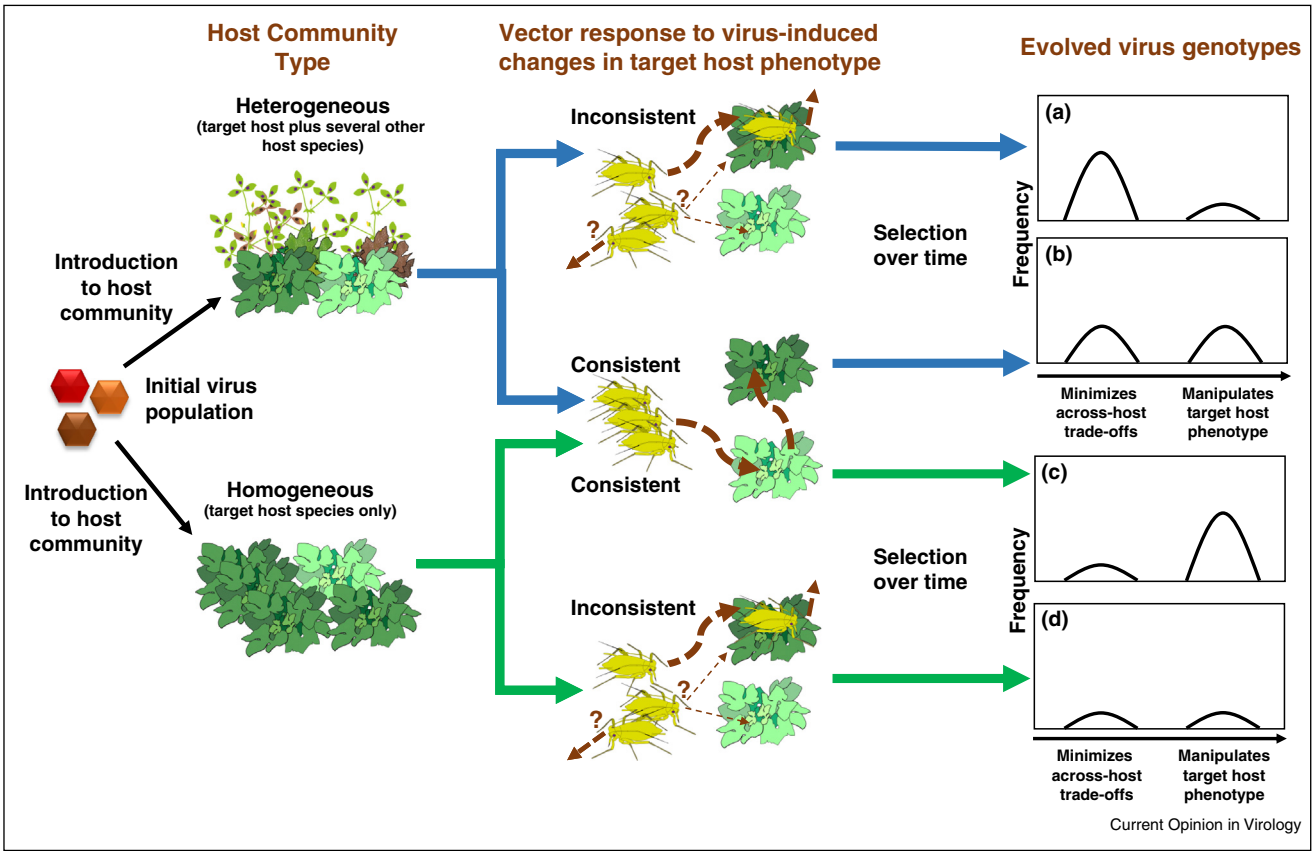
Summary of studies describing changes in the magnitude or sign of virus effects due to intraspecific or interspecific variation in the host plant or vector

Transmission mechanism	Virus	Sources of variation	Variation associated with virus infectivity or severity of infection?	References
Non-circulative Non-persistent	*Cucumber mosaic virus* (CMV) Bromoviridae	Host species, host novelty for virus	Yes	[41••]
		Disease progression	Yes	[48]
		Host species	Untested	[71]
	Potato virus Y (PVY) Potyviridae	Host species	Untested	[72]
	Zucchini yellow mosaic virus (ZYMV) Potyviridae	Disease progression	Untested	[73]
	Turnip mosaic virus (TuMV) Potyviridae	Host cultivar Vector species Disease progression	Yes	[74]
	Sweet potato feathery mottle virus (SPFMV) Potyviridae	Host species	Yes	[42,43]
Non-circulative Semi-persistent	*Beet yellows virus* (BYV) Closteroviridae	Virus genotype Vectors' previous host plant	Yes	[75]
Circulative Persistent Non-propagative	*Potato leafroll virus* (PLRV) Luteoviridae	Host cultivar	Yes	[36••]
		Disease progression	Yes	[45,47]
		Age of inoculation	Yes	[46••,47]
	Barley yellow dwarf virus (BYDV) Luteoviridae	Host cultivar	Yes	[37–39]
		Host species	Yes *Severity of symptoms associated with greater attraction of vectors*	[76]
		Host cultivar	No *Effects vary with aphid resistance level of each cultivar*	[77]
	Tomato yellow leaf curl virus (TYLCV) and *Tomato yellow leaf curl China virus* (TYLCCV) Geminiviridae	Host cultivar	Yes	[10]
		Vector biotype	Unknown *Possible direct effects of virus circulating in vectors*	[78–81]
	Squash leaf curl virus (SLCV) Geminiviridae	Host species	Unknown	[82]
Circulative Persistent Propagative	*Tomato spotted wilt virus* (TSWV) Bunyaviridae	Host species	Unknown *Magnitude of effects varies with quality of each species for vector*	[83]
		Host species Host genotype	Unknown *Magnitude of effects varies with quality of each species for vector*	[84]
		Host species Vector species Virus genotype Temperature	Unknown	[85,86]
	Maize mosaic virus (MSV) Rhabdoviridae	Age of inoculation Disease progression	Yes *Severity of symptoms associated with greatest effects*	[87]

Potential constraints on the evolution of 'manipulative' viruses

Viruses can evolve increased infectivity or multiplication rates when repeatedly passed through one plant species or genotype, with the cost of lower infectivity or multiplication rates in other (novel) plants [23–27,28•]. These fitness costs can become more pronounced as phylogenetic distance between the original plant host and the novel host increases [29•]. Virus specialization on a host plant occurs via several non-exclusive mechanisms. Under one mechanism, called *antagonistic pleiotropy*, a virus accumulates mutations that are beneficial in the local host but detrimental in a novel host (reviewed in [24,25,30••]). Mutational effects are also typically not additive, but interactive: the effect of one mutation can depend on the presence of a second mutation, and vice versa — a mechanism known as *epistasis*. Epistasis can limit the range of adaptations available to plant viruses by making it difficult to transition from one 'adaptive peak' to the next because this shift requires the virus to exist for some period of time in a 'fitness valley' (mutation combination with lower titer or infectivity) [31–33]. Epistatic effects

Figure 2



Hypothesized effects of host and vector communities on the evolution of manipulative virus genotypes. In these hypothetical scenarios, the extremes of different plant and vector communities are portrayed and virus effects are examined in terms of a single host plant species (the target host), as is typically done in empirical studies exploring virus effects on host phenotype and vector behavior. The host plant community can be heterogeneous in terms of interspecific or intraspecific variation, or homogeneous (as in monocultures). Within each plant community, vectors can be more or less consistent in how they respond to virus effects on plant phenotypes. In heterogeneous plant communities, where there are higher costs for specializing, viruses are expected to maximize fixation of mutations that make them better generalists. As a result, selection should disfavor traits that permit host-specific phenotype manipulations (panel A), unless vector responses are very consistent and favor movement between certain hosts over others (i.e. the target host pictured here) (panel B). In a homogeneous plant community, where costs of specializing are low, selection will favor mutations that enhance fitness within the uniform host plant environment at the expense of mutations that enhance fitness across multiple host plant environments (panels C and D showing no selection for adaptations that minimize across-host trade-offs). If vector responses to virus effects on host phenotype are consistent, this will favor manipulative genotypes (panel C). If vector responses are inconsistent, manipulative genotypes will not increase in frequency (panel D) and selection will favor viruses that maximize infectivity or titer in the one host available. Neutral virus genotypes (those causing no shift in phenotype or a shift that does not result in altered vector behavior) are also expected to persist in the population, while genotypes causing a reduction in the likelihood of virus transmission via changes to host phenotypes should be selected against. This graphic provides one possible explanation for why there is substantial variation in virus effects on host phenotype and vector behavior in empirical studies, since effects will depend on the evolutionary history of a virus with a given host and vector combination. Other factors that could alter host phenotype (and expression of virus effects) include abiotic environmental effects, other plant-associated microbes, host age, plant interactions with non-vector herbivores, vector–predator interactions, and vector abundance.

can also be pleiotropic [29•]. That is, the effect of one mutation on the other (magnitude or sign-positive or negative) depends on the host plant genotype or species. When viruses induce a transmission-conducive phenotype in one host and a detrimental phenotype in another, this effect could also be due to antagonistic pleiotropy or epistatic pleiotropy. If virus effects on plant phenotypes are constrained by one or both of these mechanisms, then natural selection for or against 'manipulative' genotypes should be shaped, at least in part, by the degree of heterogeneity in the plant community coupled with the consistency of vector behavioral responses (Figure 2) [24].

The influence of host identity on virus-induced changes in host phenotype

Studies of virus effects on plant phenotype have largely focused on single virus–plant–vector combinations examined under controlled conditions, and almost never consider the plant backgrounds in which a virus has evolved

[3,4•,34]. Recent work has begun to address this discrepancy by examining virus effects under both intraspecific and interspecific host plant variation (Table 1). These studies support the hypothesis that virus effects on plant phenotype can be pleiotropic. Furthermore, when pleiotropic effects are apparent, there are often positive correlations between transmission-conducive plant phenotypes and other measures of *within-plant fitness* (infectivity, titer, or systemic spread). For example, infection of potato by the circulative virus, *Potato leafroll virus* (PLRV), renders plants more attractive (via volatiles) to a key aphid vector, *Myzus persicae* [6,35]. However, this effect only occurs in cultivars that are more easily infected by PLRV [36••]. Earlier studies with the related circulative virus, *Barley yellow dwarf virus* (BYDV) found that wheat plants with low BYDV titers exhibited transmission-limiting phenotypes, but those with high titers exhibited transmission-enhancing phenotypes [37–39]. And in a very different system involving a whitefly-transmitted circulative virus (*Tomato yellow leaf curl virus* [TYLCV]) beneficial effects on vector settling and performance were more pronounced for susceptible tomato cultivars that also had higher titers of the virus [10].

Interspecific host plant variation might be expected to have even stronger effects on the magnitude or sign of virus-induced changes in host phenotypes because fitness burdens are often greater during interspecific host jumps [24,25,40]. This was the case in an explicit test of local adaptation performed with two isolates of the non-circulative virus, *Cucumber mosaic virus* (CMV), originating from different plant communities (monocultures of squash vs. pepper). Each isolate induced changes in volatile cues and palatability in its native host plant that were conducive to virus transmission by aphids [41••]. But in a novel host, isolates either did not infect, or induced a transmission-limiting phenotype. This phenotype was also associated with lower within-plant fitness relative to virus infections in native hosts [41••]. And in another non-circulative virus system, more transmission-conducive phenotypes were only observed in *Ipomoea* species that supported high titers of *Sweet potato feathery mottle virus* (SPFMV) [42,43].

These studies, and several others (Table 1), provide evidence of pleiotropy in virus effects on plant phenotype and vector behavior across host plant environments while exposing positive correlations between transmission-conducive effects and greater within-host fitness or virulence. The observed pleiotropy can be more or less antagonistic depending on whether the phenotypes induced in alternate host plants are neutral or detrimental for transmission (Table 1). These findings are consistent with the general observation of antagonistic pleiotropy and/or epistatic pleiotropy constraining the ability of viruses to be equally fit in all possible hosts [24], but are not sufficient to demonstrate that transmission-conducive effects are the result of specific viral adaptations (Figure 2) because these effects often correlate with the severity of infection.

Variation in virus effects across host developmental stages

Virus within-plant fitness also varies with plant phenology [44]. Thus, another way to examine the relationship between virus effects and within-plant fitness is to determine how these effects change with host development. A few studies have taken this approach (Table 1). In the PLRV-potato system, attraction of aphids to volatiles of infected potatoes depends on both the time since inoculation (disease progression) [45] and the developmental stage of the plant at inoculation [46••]. In both cases, effects are putatively linked to age-related variation in PLRV susceptibility: potatoes at younger stages are more susceptible to PLRV infection and systemic spread [47] and show more transmission-conducive phenotype shifts when infected [45,46••]. A relationship between within-host fitness and the sign of virus effects (positive or negative) has also been shown for CMV [48]. Infection in tobacco enhances palatability and quality for vectors at early stages of infection when CMV titer is low — an effect that hinders transmission of this non-circulative virus by inducing aphid arrestment (Figure 1). However, just a few days later, once virus titer is at maximum, plants become unpalatable, low-quality hosts for vectors and also stimulate production of winged offspring, which are better dispersers [48]. The phenotypic changes with disease progression described here seem to be beneficial for their respective pathogens because they first enhance the probability of virus acquisition and later, inoculation [7] (Figure 1). But they also track within-plant fitness, further supporting a connection between pathology and the magnitude or sign of virus effects on the probability of vector transmission.

Implications for the evolution of 'manipulative' viruses

Although most of the reports discussed above and highlighted in Table 1 are not explicit tests of virus adaptation for manipulating specific plant hosts (but see [41••]), they do suggest a link between transmission-conducive virus effects and other within-plant fitness traits that are expected to be under stronger selection pressure [25]. Based on this finding, it is possible to derive specific hypotheses regarding the mechanisms underlying this apparent relationship, which are described in detail in Box 1. These hypotheses can serve as a framework for future studies focused on discriminating adaptive effects from by-products of infection, testing for local adaptation in virus effects on plant phenotype, and determining the relevance of virus effects for the spread of disease in agricultural and natural landscapes.

The hypotheses presented in Box 1 are also excellent starting points for incorporating the vector community

Box 1 Potential mechanisms underlying variation in virus effects on host phenotypes and vector behavior

Hypothesis 1. Virus-induced changes in host phenotype are by-products of infection. Apparently adaptive patterns of transmission-mechanism specific, neutral to beneficial host phenotype changes (Figure 1) could reflect sampling biases toward agriculturally relevant viral pathogens that have been selected for high pathogenicity in agricultural landscapes. These viruses have typically been the focus of most plant virology research to date, including observations of virus effects on host phenotypes and vector behavior. Under this hypothesis, effects on host phenotype reflect the relative infectivity and/or replication rates of highly virulent pathogens across host genotypes or species.

Hypothesis 2. Mutations that result in 'manipulative' virus genotypes are epistatically linked to traits conferring infectivity or pathogenicity in local hosts, and so are selected together during the process of local adaptation. Mutations for manipulating plant phenotype are somewhat analogous to mutations that affect vector transmissibility in that the beneficial effects of such mutations are (theoretically) only realized during movement between plants, with potentially weak selection for maintenance within a plant. Nonetheless, vector transmissibility is rarely lost during within-plant amplification and systemic spread, probably owing to the fact that traits conferring vector transmissibility can also influence infectivity, replication, and expression of symptoms [67,68]. This may also be the case for traits that affect plant phenotype. If mutations conferring manipulative ability have epistasis with mutations that are favored during local adaptation, then the sign (positive or negative) or magnitude of these mutations could change in new host plants (epistatic pleiotropy), creating an apparent link between virus within-host fitness and manipulative effects.

Hypothesis 3. Hosts that are highly susceptible to viruses exhibit more pronounced, transmission-conducive phenotypic shifts when infected relative to more resistant hosts. This hypothesis is derived from the finding that the best virus reservoirs are short-lived hosts that tend to have poor immune defenses, high nutrient levels, and high metabolisms — features that make them susceptible to both pathogen infection and vector attack [69,70]. These features are typical of the annual crop hosts in which most virus effects have been observed. Such features may be modified during breeding for virus resistance, resulting in an apparent correlation between lower titer and less transmission-conducive plant phenotypic shifts under virus infection.

into the adaptive landscape. Ultimately, the fitness outcome of any virus effect on plant phenotype will depend on whether this phenotype consistently elicits a positive response by vectors (Figure 2). It is now apparent that two or more vector genotypes or species can have divergent responses to the same suite of virus-induced phenotype changes (Table 1). This can have important implications for the evolution of manipulative virus genotypes since many viruses are transmitted by more than one vector species, each of which can vary substantially in preferences, competence (transmission ability) and efficiency (likelihood of transmitting). Thus, we might expect to detect more instances of host phenotype manipulation among virus species that have tightly co-evolved relationships with only one or two vectors (e.g. many circulative viruses), and more variable phenotypic effects of viruses that are transmitted by several vector species [49]. For example, many non-circulative viruses are spread by a large number of non-colonizing vectors that already perform the rapid probing and dispersal behaviors necessary for transmission [50]. Given that most of these viruses are also multi-host pathogens, many transmission events are mediated by non-colonizing vectors that disperse readily after perceiving taste cues of an unsuitable (but infected) host plant species. This frequent transmission by non-colonizing vectors could weaken selection pressure for viruses to influence the probing behavior of *colonizing* vectors via changes to plant phenotype. Instead, selection may favor virus genotypes that induce plant phenotypes which are broadly attractive to vectors regardless of their colonizing status (e.g. yellow coloration or enhanced volatile emissions), as this will increase recruitment of both colonizing and non-colonizing species.

Conclusions

Teasing apart the different selection pressures and testing the proposed hypotheses (Box 1) will require movement away from work with laboratory strains and toward a renewed consideration of the contexts in which viral pathogens evolve. For example, testing for local adaptation (Box 1, Hypothesis 2) involves crossing multiple field-collected virus isolates with multiple populations (or species) of host plants, then examining changes in virus replication, plant phenotypes, and vector behavior using a common set of experimental approaches [51•,52,53•]. Ideally, such studies would also consider the past history of a virus isolate with a given plant [34], plant phylogenetic relatedness [29•], and plant susceptibility [28•]. And discriminating by-products of infection from adaptations (Box 1, Hypothesis 1) will require combining these approaches with virus sequencing [28•] or examination of effects on different plant phenotypes following experimental evolution, targeted mutagenesis of viral genes, or transgenic expression of virus genes in plants [53•,54,55•,56••]. These are well-established approaches for elucidating the molecular mechanisms underlying virus evolution and adaptation to plant hosts, but are not routinely incorporated into studies on virus-induced changes in host phenotype (but see [56••]). Furthermore, these approaches have not incorporated vector transmission, instead relying on mechanical passage of large numbers of virions or the use of infectious clones. Thus, combining experimental evolution with studies of virus effects on host phenotype and vector behavior will require an integrated approach that unites the fields of virology and entomology.

Beyond mechanistic studies, it is also essential to determine if the differential suitability of pathogen infected vs. healthy hosts for vectors actually influences pathogen spread in real-world plant communities. For example, extensive research on the dynamics of infection and co-infection of wild grasses by diverse strains of BYDV

has provided conflicting reports on the role of vector preferences in driving virus prevalence. Some studies suggest that host suitability for vectors does play a role in overall disease spread [57–59] while other studies report no connection between host suitability for vectors in the laboratory and importance of that host for disease spread in the field [60,61]. However, these studies only assessed the relative suitability of wild hosts for vectors using healthy plants and did not examine how infection might alter host suitability or attractiveness to vectors [59]. Nonetheless, the BYDV-grassland system, as well as other recently described wild plant pathosystems (e.g. viruses of wild cucurbits [62–64]), are promising systems for future studies that explore links between virus-induced changes in host phenotypes, vector behavior, and the incidence of host species and virus genotypes within wild plant communities. Although expanding the context in which we study virus effects on host-vector interactions will be challenging, it is a necessary step on the path to a deeper understanding of plant virus evolution and adaptation.

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Using experimental evolution of an isolate of *Tobacco etch virus* in different genotypes of *Arabidopsis thaliana*, this study demonstrates that local adaptation to a specific plant genotype does not always result in reduced fitness in the original host plant. Whether an evolved virus lineage is more specialist or more generalist depends on the host genotype in which it was passaged. Furthermore, this study shows that virus titer is not always linked to the expression of virulent symptoms, but also depends on the specific plant–virus combination. This evolutionary approach could be applied to the study of virus effects on host plant phenotypes.

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This study examines the magnitude and sign of epistasis across phylogenetically divergent host plants for *Tobacco etch virus* genotypes that each contain two different mutations with known independent effects. Across virus genotypes, the degree of host effect on epistasis is influenced by genetic divergence between the novel host and the original host.

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This short review discusses the relative importance of antagonistic pleiotropy, epistasis, and epistatic pleiotropy for the emergence of viral pathogens in new host communities. In addition to being an excellent primer on these topics, it emphasizes the need to integrate environmental effects (host plant age, biotic stressors, and vector physiological condition) into studies of virus adaptation to novel host plants.

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This study examines virus-induced changes in host plant cues across different genetic backgrounds of the same host species (potato) that also vary in susceptibility to virus infection. More resistant cultivars showed few changes in blend composition or concentration, while susceptible cultivars showed divergent changes (compound concentrations vs. compound composition) that both resulted in increased aphid arrestment via different mechanisms. This result demonstrates that even transmission-conducive virus effects can be expressed as different cues depending on host genetic background.

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In this study, transmission-conducive virus effects were only apparent if plants became infected at early developmental stages. Plants inoculated at later stages exhibited transmission-limiting effects. Thus the sign of virus effects changed depending on plant developmental parameters. These effects are also putatively linked to the ability of the virus to exploit different host plant developmental stages, which become increasingly more resistant to infection throughout developmental progression.

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By evolving *Pepino mosaic virus* on three tomato hosts that differed in susceptibility to the virus, researchers were able to track the emergence of various symptoms alongside the experimental evolution of the pathogen. Notably, the same symptoms appeared in multiple, replicate lineages within the same host plant genotype, demonstrating the importance of the host environment for the evolution of virus effects on host phenotype.

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Following up on a suite of studies exploring the evolution of *Tobacco etch virus* (TEV) across different *Arabidopsis thaliana* ecotypes, this study characterizes gene expression in each ecotype when infected with an ancestral isolate of TEV, and different lineages of TEV evolved towards generalist or specialist life styles. The results are consistent with the hypothesis that resistance genes are not the only factors shaping virus evolution on a host because specialist and generalist TEV variants had divergent effects on the transcriptomes of the same ecotypes.

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This study explores the hypothesis that viral suppressors of RNA silencing (VSRs) are the primary inducers of phenotypic changes in host plants. The authors examine changes to host phenotype (jasmonic acid-mediated vector defenses) during transgenic expression of VSRs in host plants and compare this to infection with the wild-type virus. While VSRs do influence the expression of JA-based defenses in similar ways across diverse virus species, this does not always result in the same effects on host-vector interactions, suggesting that epistatic effects are important for induction of host phenotype shifts.

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